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NEWS 22 Aug 26 Sequence searching in REGISTRY enhanced  
NEWS 23 Sep 03 JAPIO has been reloaded and enhanced  
NEWS 24 Sep 16 Experimental properties added to the REGISTRY file  
NEWS 25 Sep 16 Indexing added to some pre-1967 records in CA/CAPLUS  
NEWS 26 Sep 16 CA Section Thesaurus available in CAPLUS and CA  
NEWS 27 Oct 01 CASREACT Enriched with Reactions from 1907 to 1985  
NEWS 28 Oct 21 EVENTLINE has been reloaded  
NEWS 29 Oct 24 BEILSTEIN adds new search fields  
NEWS 30 Oct 24 Nutraceuticals International (NUTRACEUT) now available on STN  
NEWS 31 Oct 25 MEDLINE SDI run of October 8, 2002  
  
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=> s ischemia (5A) actin

35 FILES SEARCHED...

81 FILES SEARCHED...

L1 244 ISCHEMIA (5A) ACTIN

=> s actin (5A) (caudate or cortex)

35 FILES SEARCHED...

62 FILES SEARCHED...

L2 1753 ACTIN (5A) (CAUDATE OR CORTEX)

=> s actin (5A) (substantia nigra)

32 FILES SEARCHED...

66 FILES SEARCHED...

L3 6 ACTIN (5A) (SUBSTANTIA NIGRA)

=> s l2 or l3

53 FILES SEARCHED...

L4 1757 L2 OR L3

=> s l1 and l4

55 FILES SEARCHED...

L5 0 L1 AND L4

=> s size (4A) (cell or neuron)

9 FILES SEARCHED...

13 FILES SEARCHED...

26 FILES SEARCHED...

30 FILES SEARCHED...

43 FILES SEARCHED...

53 FILES SEARCHED...

55 FILES SEARCHED...

69 FILES SEARCHED...

L6 214897 SIZE (4A) (CELL OR NEURON)

=> s ischemia (4A) 16

51 FILES SEARCHED...

L7 14 ISCHEMIA (4A) L6

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AN 2001:32482 BIOSIS

DN PREV200100032482

TI Treatment with nimodipine or mannitol reduces programmed cell

L4 ? Ischemia

death and infarct size following focal cerebral ischemia

AU Korenkov, Alexei I. (1); Pahnke, Jens; Frei, Karl; Warzok, Rolf; Schroeder, Henry W. S.; Frick, Rosemarie; Muljana, Lydia; Piek, Juergen; Yonekawa, Yasuhiro; Gaab, Michael R.  
CS (1) Department of Neurosurgery, University of Greifswald, Sauerbruchstrasse, 17487, Greifswald: korenkow@mail.uni-greifswald.de Germany  
SO Neurosurgical Review, (September, 2000) Vol. 23, No. 3, pp. 145-150. print.  
ISSN: 0344-5607.  
DT Article  
LA English  
SL English  
AB The present study was conducted to evaluate the effects of nimodipine and mannitol on infarct size and on the amount of apoptosis after transient focal cerebral ischemia. Focal cerebral ischemia was induced in male Sprague-Dawley rats (weight 300-380 g) by transient occlusion of the right middle cerebral artery (MCAO) using an intraluminal thread model. All animals underwent ischemia for 2 h, followed by 24 h of reperfusion. Group I (n=16) was untreated. Group II (n=16) received 15% mannitol (1 g/kg as bolus) and group III (n=9) received 15 mug/kg/h nimodipine intravenously beginning 15 min prior to MCAO. Twenty-four hours after reperfusion, the brain was taken and sectioned in coronal slices. The slices were stained with H&E and with the transferase dUTP nick-end labeling (TUNEL) technique. Histopathological analysis revealed a significant ( $P<0.05$ ) decrease in infarct size in the striatum with both drugs: mannitol (group II) 25.4+-5.9% and nimodipine (group III) 21.5+-11.0% versus control (group I) 34.9+-7.0% and in the cortex 2.7+-2.0% (group II) and 6.3+-2.4% (group III) versus control 14.4+-9.0% (group I). The number of apoptotic cells was statistically lower in the therapy groups (group III 9.6, group II 25.8) versus control (group I 57.9) (Mann-Whitney-Wilcoxon U-test  $Z>1.96$ ,  $P<0.05$ ). This study indicates that mannitol and nimodipine provide neuroprotection by preventing both the necrotic and apoptotic components of cell death after transient focal cerebral ischemia and may be effective as neuroprotective drugs for cerebrovascular surgery.

L8 ANSWER 2 OF 8 JICST-EPlus COPYRIGHT 2002 JST  
AN 1000629796 JICST-EPlus  
TI Melatonin Protects Against Ischemia-Reperfusion Induced Astrocytic Cell Death and Reduces the Size of the Infarction.  
AU KUMAZAKI M; YAMAMOTO M; TAKEI N; HIDA H; NISHINO H  
CS Nagoya City Univ. Medical School, Aichi, Jpn  
SO Jpn J Physiol, (1999) vol. 49, no. Supplement, pp. S235. Journal Code: Z0753A  
CODEN: JJPHAM; ISSN: 0021-521X  
CY Japan  
LA English  
STA New

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AN 1998:883998 SCISEARCH  
GA The Genuine Article (R) Number: 131UV  
TI Effect of caspase-1 or caspase-3 inhibitor on infarct size and apoptotic cell death in the ischemia reperfused rat heart  
AU Okamura T (Reprint); Miura T; Iwamoto H; Shirakawa K; Kawamura S; Ikeda Y; Iwatate M; Matsuzaki M  
CS YAMAGUCHI UNIV, UBE, YAMAGUCHI 755, JAPAN  
CYA JAPAN  
SO CIRCULATION, (27 OCT 1998) Vol. 98, No. 17, Supp. [S], pp. 1353-1353. Publisher: WILLIAMS & WILKINS, 351 WEST CAMDEN ST, BALTIMORE, MD 21201-2436.

ISSN: 0009-7322.  
DT Conference; Journal  
FS LIFE; CLIN  
LA English  
REC Reference Count: 0

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AN 1999:523828 BIOSIS  
DN PREV199900523828  
TI Effect of caspase-1 or caspase-3 inhibitor on infarct size and apoptotic cell death in the ischemia-reperfused rat heart.  
AU Okamura, Takayuki (1); Miura, Toshiro (1); Iwamoto, Hiroshi (1); Shirakawa, Kazuyuki (1); Kawamura, Shuji (1); Ikeda, Yasuhiro; Iwatate, Mitsuo  
CS (1) Yamaguchi Univ., Ube Japan  
SO Circulation, (Oct. 27, 1998) Vol. 98, No. 17 SUPPL., pp. I260.  
Meeting Info.: 71st Scientific Sessions of the American Heart Association Dallas, Texas, USA November 8-11, 1998 The American Heart Association . ISSN: 0009-7322.  
DT Conference  
LA English

L8 ANSWER 5 OF 8 CAPLUS COPYRIGHT 2002 ACS DUPLICATE 2  
AN 1997:481810 CAPLUS  
DN 127:170895  
TI Selfotel (CGS19755)  
AU Schmutz, Markus; Arthur, A.; Faleck, H.; Karlsson, G.; Kotake, A.; Lantwicki, L.; Larue, L.; Markabi, S.; Murphy, D.; Powell, M.; Sauer, D.  
CS Research & Development, Pharmaceuticals Division, Ciba, Basel, CH-4002, Switz.  
SO Excitatory Amino Acids: Clinical Results with Antagonists (1997), 1-6, 129-152. Editor(s): Herrling, P. L. Publisher: Academic, London, UK.  
CODEN: 64UIAO  
DT Conference; General Review  
LA English  
AB A review with over 550 refs. Selfotel (CGS19755) is a potent selective and competitive N-methyl-D-aspartate (NMDA) antagonist. Preclinically, selfotel reduced ischemia-induced infarct size and neuronal cell death, antagonized the effects of excitotoxic lesions in the brain, and attenuated neuronal damage following traumatic brain injury at i.p. or i.v. doses ranging from 3 to 40 mg kg<sup>-1</sup>. In addn. to these neuroprotectant properties, selfotel also exhibited anticonvulsant and anxiolytic activity. Behavioral central nervous system (CNS) effects include ataxia and increased locomotor activity. The compd. was not mutagenic, clastogenic, or teratogenic in rats or rabbits. Similar to other competitive and noncompetitive NMDA antagonists, selfotel produced Olney-type vacuoles in a dose-related manner in rat brain. At present, the clin. significance of these findings in rats is unknown. In humans, the plasma pharmacokinetics of selfotel were linear in the dose range evaluated, and elimination half-life, clearance and vol. of distribution at steady state were independent of dose. There was no appreciable protein binding. Preliminary human data indicate that selfotel rapidly crosses the blood-brain barrier and remains in the cerebral spinal fluid (CSF) for an extended period of time. Doses of up to 3 mg kg<sup>-1</sup> i.v. have been evaluated in conscious healthy male subjects, with non-psychotomimetic CNS adverse experiences being the dose-limiting factors. Administration of single doses of up to 2 mg kg<sup>-1</sup> of selfotel did not impact the management of neurosurgical patients. However, a no. of these patients also experienced CNS effects. In patients who were conscious following an acute ischemic stroke, doses up to an including 1.5 mg kg<sup>-1</sup> i.v. were found safe and were tolerated. Dose-limiting adverse experiences in conscious patients included transient agitation, hallucinations, and confusion at higher doses. In unconscious patients

treated with selfotel following severe closed traumatic brain injury, four bolus doses of up to 5 mg kg<sup>-1</sup>, administered over a 72 h period, appeared safe and well tolerated. Four international well-controlled clin. trials including approx. 3600 patients, two trials each in acute ischemic stroke and severe traumatic brain injury, are currently underway to evaluate the definitive safety and efficacy of selfotel in improving the functional outcome of patients with these disorders.

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AN 1995:428846 BIOSIS  
DN PREV199598443146  
TI Recombinant colony stimulating factor-1 reduces infarct size and rescues neurons in cerebral focal ischemia.  
AU Berezovskaya, O. (1); Maysinger, D.; Zhai, R. (1); Fedoroff, S.  
CS (1) Dep. Pharmacol., Univ. Saskatchewan, Saskatoon, SK S7N 5E5 Canada  
SO Society for Neuroscience Abstracts, (1995) Vol. 21, No. 1-3, pp. 540.  
Meeting Info.: 25th Annual Meeting of the Society for Neuroscience San Diego, California, USA November 11-16, 1995  
ISSN: 0190-5295.  
DT Conference  
LA English

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AN 1988:73184 CAPLUS  
DN 108:73184  
TI Dopamine depletion protects striatal neurons from ischemia-induced cell death  
AU Clemens, James A.; Phebus, Lee A.  
CS Lilly Res. Lab., Lilly Corp. Cent., Indianapolis, IN, 46285, USA  
SO Life Sciences (1988), 42(6), 707-13  
CODEN: LIFSAK; ISSN: 0024-3205  
DT Journal  
LA English  
AB Infusion of the dopaminergic neurotoxin 6-hydroxydopamine, unilaterally into the substantia nigra of rats, resulted in a unilateral depletion of dopamine in the ipsilateral brain striatum. When these rats were subjected to global forebrain, ischemia, there was a marked protection of the dopamine-depleted striatum from the ischemia-induced loss of medium size neurons seen in the intact striatum. These results imply a role for dopamine in the ischemia-induced striatal cell death.

L8 ANSWER 8 OF 8 CAPLUS COPYRIGHT 2002 ACS  
AN 1976:521268 CAPLUS  
DN 85:121268  
TI Quantitative oxidative enzyme histochemistry of the spinal cord. Part 2. Relation of cell size and enzyme activity to vulnerability to ischaemia  
AU Penny, J. E.; Kukums, J. R.; Tyrer, J. H.; Eadie, M. J.  
CS Dep. Med., Univ. Queensland, Brisbane, Aust.  
SO J. Neurol. Sci. (1975), 26(2), 187-92  
CODEN: JNSCAG  
DT Journal  
LA English  
AB Cytophotometric measurements of the activities of 5 enzymes (succinate, malate, and NAD-linked isocitrate dehydrogenases; lactate dehydrogenase, and NADH dehydrogenase) were correlated with cell vol. for neurons in the anterior horn of rabbit lumbar and cervical spinal cord. The data for succinate and isocitrate dehydrogenases indicated that these enzymes were at higher concns. in the smaller neurons, which consist largely of interneurons. No preferential localization to particular sizes of cell could be assigned to the other enzymes studied. The relation between enzyme distribution patterns and their possible role in contributing toward susceptibility to ischemia of particular sizes of neurons is discussed.

=>

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L10 ANSWER 1 OF 6 USPATFULL  
AN 2002:164662 USPATFULL  
TI System and method for investigating the effect of chemical and other  
factors on cell movement  
IN Lynes, Michael A., Willington, CT, UNITED STATES  
Knecht, David A., Storrs, CT, UNITED STATES  
PI US 2002086280 A1 20020704  
AI US 2001-2961 A1 20011026 (10)

PRAI US 2000-243450P 20001026 (60)  
DT Utility  
FS APPLICATION  
LREP Cummings & Lockwood, P.O. Box 1960, New Haven, CT, 06509-1960  
CLMN Number of Claims: 20  
ECL Exemplary Claim: 1  
DRWN 14 Drawing Page(s)  
LN.CNT 1253

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB As disclosed herein, the present invention is directed to a novel system for monitoring cell movement in response to chemotactic and chemokinetic factors. In this system, cells migrate in an under-agarose environment and their position is monitored using a system capable of measuring changes in impedance and other electrical parameters of the system at a target electrode lithographed onto a substrate as the cells arrive at the target. With the disclosed system, the time of arrival of cells at the target electrode is proportional to the dose of the chemoattractant species used to stimulate the cells and can be assessed by changes in resistance at the electrode. The system is readily able to distinguish between wild-type cells and mutants that are deficient in their chemotactic response. In addition, agents that interfere with chemotactic motility can be shown to lead to delayed arrival of cells at the target electrode. The multi-well configuration of the disclosed assay system allows for simultaneous automated screening of many samples for chemotactic or anti-chemotactic activity.

=> d 110 1-6 bib ab

L10 ANSWER 1 OF 6 USPATFULL  
AN 2002:164662 USPATFULL  
TI System and method for investigating the effect of chemical and other factors on cell movement  
IN Lynes, Michael A., Willington, CT, UNITED STATES  
Knecht, David A., Storrs, CT, UNITED STATES  
PI US 2002086280 A1 20020704  
AI US 2001-2961 A1 20011026 (10)  
PRAI US 2000-243450P 20001026 (60)  
DT Utility  
FS APPLICATION  
LREP Cummings & Lockwood, P.O. Box 1960, New Haven, CT, 06509-1960  
CLMN Number of Claims: 20  
ECL Exemplary Claim: 1  
DRWN 14 Drawing Page(s)  
LN.CNT 1253

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AN 2000:99857 BIOSIS  
DN PREV200000099857  
TI The use of quantitative RT-PCR to measure mRNA expression in a rat model of focal ischemia: Caspase-3 as a case study.  
AU Harrison, David C. (1); Medhurst, Andrew D.; Bond, Brian C.; Campbell, Colin A.; Davis, Robert P.; Philpott, Karen L.  
CS (1) Molecular Neurobiology Research, SmithKline Beecham Pharmaceuticals, Third Avenue, New Frontiers Science Park, Harlow, Essex, CM19 5AW UK  
SO Molecular Brain Research, (Jan. 10, 2000) Vol. 75, No. 1, pp. 143-149.  
ISSN: 0169-328X.  
DT Article  
LA English  
SL English  
AB Quantitative reverse transcription and polymerisation chain reaction (RT-PCR) using Taqman™ fluorogenic probes has been used to measure changes in gene expression in the cerebral cortex of rats in the permanent middle cerebral artery occlusion (pMCAO) model of focal ischemia. The mRNA levels of three housekeeping genes have been analysed in this model to determine which gene showed least change following experimental insult. In the lesioned cortex, beta-actin mRNA increased at 24 h, while the levels of cyclophilin and glyceraldehyde-3-phosphate dehydrogenase (GAPDH) did not change. We have also used this methodology to examine modulations in the level of caspase-3 mRNA during focal ischemia in the rat. Caspase-3 mRNA showed a 41% increase at 6 h post-MCAO, which was specific to the lesioned cortex. This change became more pronounced with time, showing an increase of 220% at 24 h. This methodology enables changes in mRNA expression to be analysed more sensitively and quantitatively than other available techniques and highlights the need for careful choice of control or housekeeping genes used for RNA comparisons.

L10 ANSWER 3 OF 6 USPATFULL  
AN 1998:150936 USPATFULL  
TI Uses of estrogen compounds for the treatment of disease  
IN Simpkins, James W., Gainesville, FL, United States  
PA University of Florida Research Foundation, Inc., Gainesville, FL, United States (U.S. corporation)  
PI US 5843934 19981201  
AI US 1996-648857 19960516 (8)  
RLI Continuation-in-part of Ser. No. US 1993-149175, filed on 5 Nov 1993, now abandoned 76 Ser. No. US 1994-318042, filed on 4 Oct 1994, now patented, Pat. No. US 5554601  
DT Utility  
FS Granted  
EXNAM Primary Examiner: Weddington, Kevin E.  
LREP Bromberg & Sunstein LLP  
CLMN Number of Claims: 6  
ECL Exemplary Claim: 1  
DRWN 11 Drawing Figure(s); 10 Drawing Page(s)  
LN.CNT 1469  
CAS INDEXING IS AVAILABLE FOR THIS PATENT.  
AB A method is provided for conferring a cytoprotective effect on a population of cells in a male or female subject which includes providing an estrogen compound having insubstantial sex related activity in a pharmaceutical formulation and administering the formulation in an effective dose to a population of cells to confer cytoprotection. The method further includes administering the estrogen compound so as to confer a cytoprotective effect in a subject so as to retard adverse effects of osteoporosis in a male or female subject.

L10 ANSWER 4 OF 6 USPATFULL  
AN 1998:54748 USPATFULL  
TI Neuron-specific ICAM-4 promoter  
IN Kilgannon, Patrick D., Bothell, WA, United States

PA Gallatin, W. Michael, Mercer Island, WA, United States  
ICOS Corporation, Bothell, WA, United States (U.S. corporation)  
PI US 5753502 19980519  
AI US 1996-656984 19960606 (8)  
RLI Continuation-in-part of Ser. No. US 1995-481130, filed on 7 Jun 1995,  
now patented, Pat. No. US 5702917 which is a continuation-in-part of  
Ser. No. US 1994-245295, filed on 18 May 1994, now patented, Pat. No. US  
5700658 which is a continuation-in-part of Ser. No. US 1993-102852,  
filed on 5 Aug 1993, now abandoned

DT Utility

FS Granted

EXNAM Primary Examiner: Ulm, John; Assistant Examiner: Brown, Karen E.

LREP Marshall, O'Toole, Gerstein, Murray & Borun

CLMN Number of Claims: 9

ECL Exemplary Claim: 1

DRWN No Drawings

LN.CNT 2173

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB DNA sequences, derived from the 5' region of the human neuron-specific  
cellular adhesion molecule ICAM-4, which promote neuron-specific gene  
transcription are disclosed along with polynucleotides comprising the  
promoter DNA operatively linked to heterologous gene-encoding  
polynucleotides, expression vectors comprising the promoter DNA, and  
host cells transformed or transfected with DNA comprising the promoter  
sequences.

L10 ANSWER 5 OF 6 USPATFULL

AN 96:82674 USPATFULL

TI Methods for neuroprotection

IN Simpkins, James W., Gainesville, FL, United States

Singh, Meharvan, Gainesville, FL, United States

Bishop, Jean, Jacksonville, FL, United States

PA University of Florida, Gainesville, FL, United States (U.S. corporation)

PI US 5554601 19960910

AI US 1994-318042 19941004 (8)

RLI Continuation-in-part of Ser. No. US 1993-149175, filed on 5 Nov 1993,  
now abandoned

DT Utility

FS Granted

EXNAM Primary Examiner: Weddington, Kevin E.

LREP Bromberg & Sunstein

CLMN Number of Claims: 29

ECL Exemplary Claim: 1

DRWN 11 Drawing Figure(s); 10 Drawing Page(s)

LN.CNT 1532

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB A method is provided for conferring neuroprotection on a population of  
cells using estrogen compounds that have insubstantial sex activity and  
furthermore, a method is provided that utilizes estrogen compounds in  
the absence of testosterone for treating neurodegenerative diseases  
including Alzheimer's disease so as to retard the adverse effects of  
these disorders. Examples of estrogen compounds that have insubstantial  
sex activity includes alpha isomers of estrogen compounds such as  
17.alpha. estradiol.

L10 ANSWER 6 OF 6 SCISEARCH COPYRIGHT 2002 ISI (R)

AN 95:22332 SCISEARCH

GA The Genuine Article (R) Number: PY085

TI TUBULIN AND ACTIN MESSENGER-RNAS IN THE YOUNG-ADULT AND THE AGED RAT-BRAIN  
- EFFECTS OF REPEATED ADMINISTRATION WITH BIFEMELANE HYDROCHLORIDE

AU NISHIBAYASHI S; OGAWA N (Reprint); ASANUMA M; KONDO Y; NORI A

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SO ARCHIVES OF GERONTOLOGY AND GERIATRICS, (NOV/DEC 1994) Vol. 19, No. 3, pp.  
265-272.  
ISSN: 0167-4943.  
DT Article; Journal  
FS LIFE  
LA ENGLISH  
REC Reference Count: 31  
\*ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS\*  
AB In an attempt to identify the age-dependent changes in the potential synthesis of cytoskeletal proteins, we investigated changes in messenger RNA (mRNA) of alpha-tubulin and beta-actin in the young-adult and the aged rat brain using Northern blot analysis. alpha-Tubulin mRNA levels in the frontal cortex and hippocampus, and beta-actin mRNA levels in the hippocampus were significantly decreased in the aged rat brain. Age-dependent decreases in these mRNAs may be related to the neuronal dysfunction associated with aging, in addition to the reduction of several kinds of receptors previously reported. Repeated administration of bifemelane hydrochloride (4-(2-benzylphenoxy)-N-methylbutylamine hydrochloride) for 14 days increased the levels of beta-actin mRNA in the frontal cortex and the striatum of both young-adult and aged rats, although the effect of bifemelane treatment was smaller and not significant in the aged group. These results suggest that bifemelane treatment may enhance the synthesis of cytoskeletal protein and promote neural plasticity by inducing neurite growth or synapse formation.

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